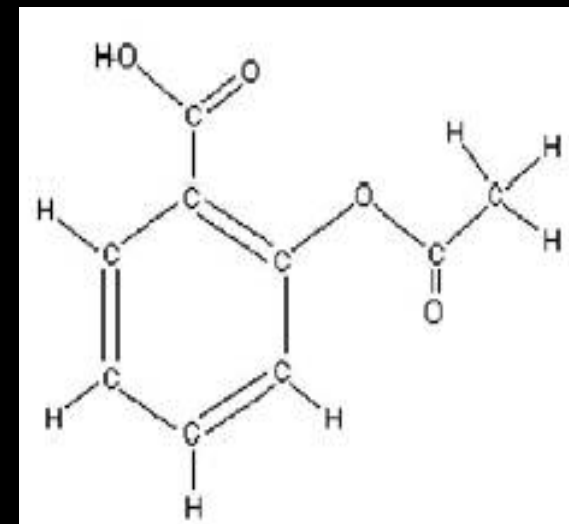
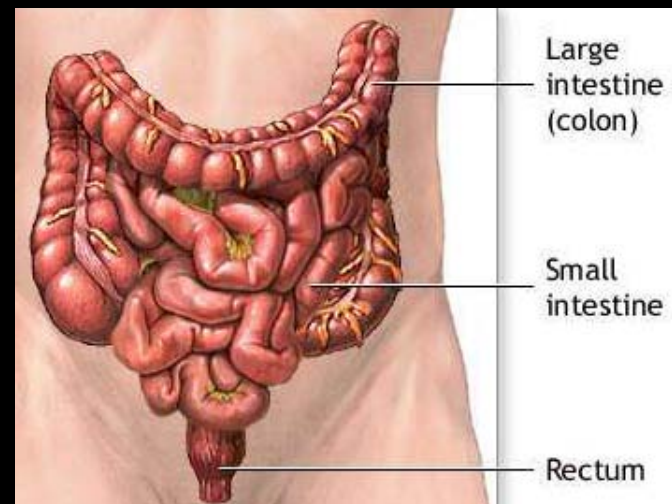
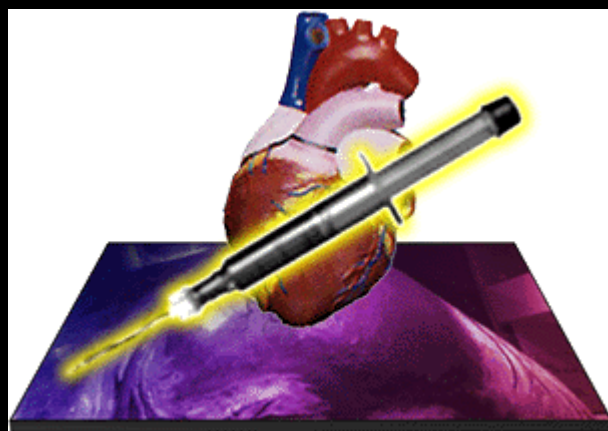




Aspirin



Could it save  
your life?



By Salina Parvez & Waqas Ali

**ASPRIN**

# Timeline of Aspirin

**c400 BC** In Greece Hippocrates gives women willow leaf tea to relieve the pain of childbirth.

**1763** Reverend Edward Stone of Chipping Norton near Oxford gives dried willow bark to 50 parishioners suffering rheumatic fever.

**1838** Salicin also found in the meadowsweet flower by Swiss and German researchers.

**1823** In Italy the active ingredient is extracted from willow and named salicin.

**1853** Salicylic acid made from salicin by French scientists but it is found to irritate the gut.

**1893** German scientists find that adding an acetyl group to salicylic acid reduces its irritant properties.

**1899** Clinical trials are successfully completed. aspirin launched.

**1897** In Germany, Bayer's Felix Hoffmann develops and patents a process for synthesising acetyl salicylic acid or aspirin. First clinical trials begin.

**1914** International trade in pharmaceuticals interrupted by the outbreak of World War I. Australian pharmacist G. R. Nicholas wins a competition to find a new way of producing aspirin.

**1930s** Bayer's patent on acetyl salicylic acid runs out. It becomes a generic drug.

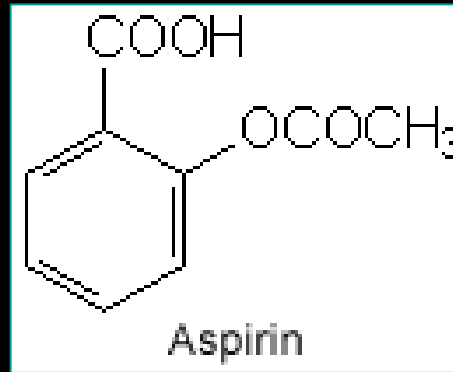
**1974** First evidence of aspirin's effects in preventing heart attacks: Professor Elwood.

**1995** American researchers find evidence that aspirin protects against bowel cancer.

**1982** English scientist Professor Sir John Vane and two Swedish colleagues, Sune Bergström and Bengt Samuelsson win Nobel prize for discovering the role of aspirin in inhibiting prostaglandin production.

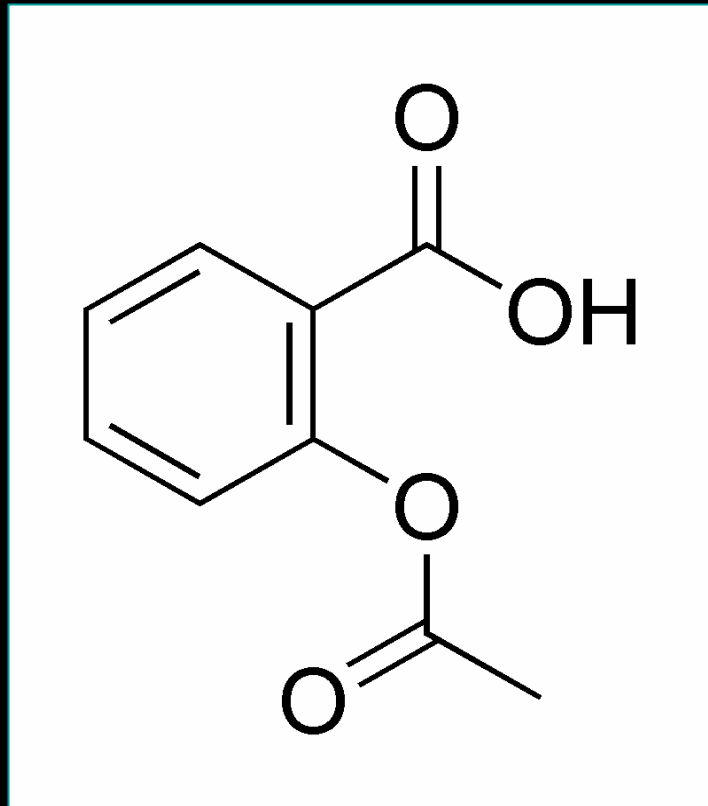
**1989** US researchers report preliminary study suggesting that aspirin may delay the onset of senile dementia  
**1994** - Professor Henk C S Wallenburg of Rotterdam shows that aspirin may help in treating pre-eclampsia in pregnant women.

# Structure of Aspirin



- Aspirin, is analgesic, anti-inflammatory, and is an inhibitor of platelet aggregation.
- Inhibits fatty acid cyclo-oxygenase by acetylation of the active site of enzyme
- Aspirin is being used for treating Cardiovascular Disease, strokes, Pregnancy Complications, lung and pancreatic cancers, diabetes and dementia .

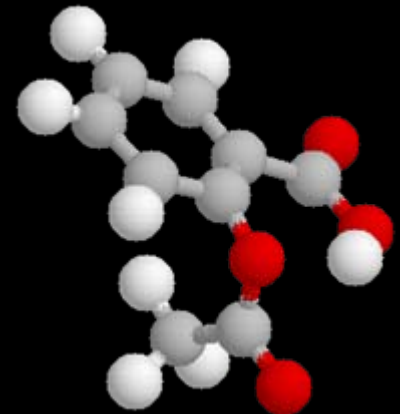
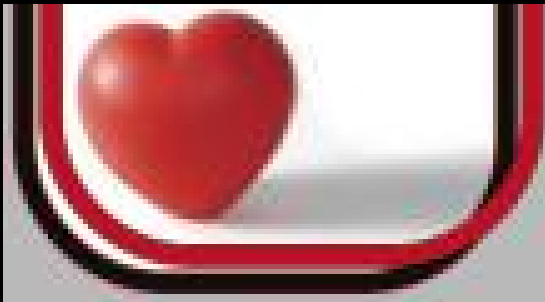
# What is Aspirin prescribed for?



PAIN  
RELIEF

# Uses of aspirin

- Pain relief, particularly where there is inflammation involved, including dental pain and period pain (dysmenorrhoea).
  - Reducing temperature (as an antipyretic).
  - Making the blood flow better through narrowed blood vessels.
- Aspirin is used to relieve mild to moderate pain; reduce fever, redness, and swelling; and to help prevent blood from clotting. It is used to relieve discomfort caused by numerous medical problems, including headache, infections, and arthritis. It is also used to reduce the risk of a second heart attack or stroke. Larger doses of aspirin are used to treat gout.



# Aspirin

Aspirin has been shown to be helpful when used daily to lower the risk of heart attack, clot-related strokes and other blood flow problems. Many medical professionals prescribe aspirin for these uses. There may be a benefit to daily aspirin use for you if you have some kind of heart or blood vessel disease, or if you have evidence of poor blood flow to the brain. However, the risks of long-term aspirin use may be greater than the benefits if there are no signs of, or risk factors for heart or blood vessel disease



**1997** aspirin is now used or being tested for use in the following conditions:-

### **Heart attacks**

Aspirin is now accepted as an important weapon in the prevention of heart disease. After the first study by Elwood and Cochrane was reported in the British Medical Journal (1974, 1, 436) larger trials involving 20,000 US doctors showed that aspirin reduced the risk of coronary thrombosis by 44 per cent. A single dose of 300 mg is now recommended for patients in the acute stages of a heart attack followed by a daily dose of 75-100 mg.

A similar low dose treatment regime is recommended for patients with angina, a history of heart problems or who have undergone coronary bypass surgery.

### **Strokes**

A trial reported in the Lancet this year (vol 349 p 1641) is the latest in a sequence of studies showing that aspirin reduces the risk of strokes in patients with 'early warning signs' of transient ischaemic attacks. Further trials showed a small but definite benefit in reducing mortality in those patients (T.I.A.'s) in the acute phase of a stroke.

### **Diabetes -**

Blindness, coronary artery disease, stroke and kidney failure are all common complications of diabetes resulting from impaired blood circulation. The benefits of taking one aspirin a day are now so widely accepted that it is considered unethical to perform placebo controlled trials to prove the case.

### **Colon cancer**

In a long term study of 90,000 US nurses between 1976 and 1995, those who took 4-6 tablets of aspirin a week had a reduced incidence of colorectal cancer. The benefits were greatest in those who had taken the drugs the longest.

# Side effects of aspirin

Most patients benefit from aspirin and other NSAIDs with few side effects. However, serious side effects can occur and generally tend to be dose related. Therefore, it is advisable to use the lowest effective dose to minimize side effects. The most common side effects of aspirin involve the gastrointestinal system and ringing in the ears. It can cause ulcerations, abdominal burning, pain, cramping, nausea, gastritis, and even serious gastrointestinal bleeding and liver toxicity. Sometimes, stomach ulceration and bleeding can occur without any abdominal pain. Black tarry stools, weakness, and dizziness upon standing may be the only signs of internal bleeding. Should ringing in the ears occur, the daily dose should be reduced. Rash, kidney impairment, vertigo, and light-headedness can also occur



# Aspirin-Mechanism of Action

- Pain is something you feel in your brain triggered by nerves throughout your body. When tissue is damaged, it creates prostaglandin, a chemical which magnifies the message to your brain sent by the nerves, making the pain felt more intense.
- Prostaglandin is made by enzymes called cyclooxygenase-2 (COX-2).
- Prostaglandin, as well as amplifying the pain signal to your brain cause swelling (inflammation) in the damaged area.
- Aspirin sticks to the enzyme that makes prostaglandins (COX-2) so that prostaglandins cannot be made. This means that the pain signal to your brain isn't amplified so that the pain felt is less intense. It also means swelling is reduced.

## Side Effects

- Aspirin doesn't just inhibit prostaglandin production at the site of pain. It stops it all over the body. This causes side effects.
- Side effects of aspirin include damage to the lining of your stomach, prolonged bleeding time, wheezing, breathlessness, ringing in the ears, hearing loss, chronic catarrh & runny nose, headache, confusion, nausea, vomiting, GI upset, GI bleeding, ulcers, rash, allergic reactions, hives, bruising, abnormal liver function tests, liver damage, and hepatitis

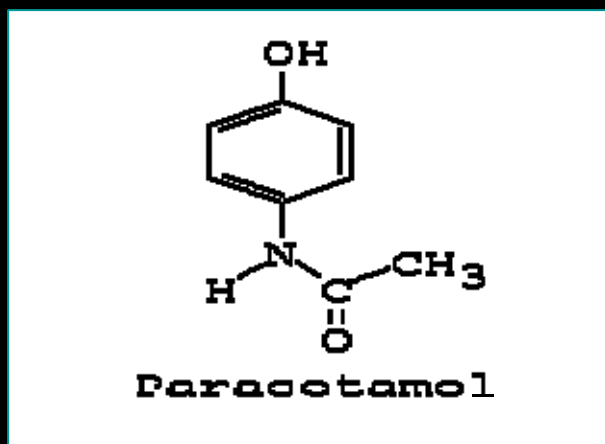


***Paracetamol***

# Structure of Paracetamol

**Paracetamol** is a common analgesic that is used for the relief of fever, headaches, and other minor aches and pains. It is a major ingredient in numerous cold and flu medications and many prescription analgesics. It is remarkably safe in standard doses, but, because of its wide availability, deliberate or accidental overdoses are not uncommon.

Paracetamol and aspirin have similar analgesic properties



# Paracetamol

In other context it is formulated as 4-hydroxyacetanilide or N-acetyl-p-aminophenol, it is a white odourless substance.

Paracetamol has long been suspected of having a similar mechanism of action to aspirin because of the similarity in structure.

Over 100 years after it was first discovered, we are now learning what the mechanism of action is that makes paracetamol such an effective and useful medicine. It now appears paracetamol has a highly targeted action in the brain, blocking an enzyme involved in the transmission of pain.



The production of prostaglandins is part of the body's inflammatory response to injury, and inhibition of prostaglandin production around the body by blocking the cyclooxygenase enzymes known as COX-1 and COX-2 has long been known to be the mechanism of action of aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen. However, their action in blocking COX-1 is known to be responsible for also causing the unwanted gastrointestinal side effects associated with these drugs. Paracetamol has no significant action on COX-1 and COX-2, which left its mode of action a mystery but did explain its lack of anti-inflammatory action and also, more importantly, its freedom from gastrointestinal side effects typical of NSAIDs.

Early work had suggested that the fever reducing action of paracetamol was due to activity in the brain while its lack of any clinically useful anti-inflammatory action was consistent with a lack of prostaglandin inhibition peripherally in the body.

Now, recent research has shown the presence of a new, previously unknown cyclooxygenase enzyme COX-3, found in the brain and spinal cord, which is selectively inhibited by paracetamol, and is distinct from the two already known cyclooxygenase enzymes COX-1 and COX-2. It is now believed that this selective inhibition of the enzyme COX-3 in the brain and spinal cord explains the effectiveness of paracetamol in relieving pain and reducing fever without having unwanted gastrointestinal side effects.

# Structure of Paracetamol

## Physical properties

Melting Point	169°C
Density	1.263 g/cm <sup>3</sup>
Solubility in water	1.4 g/100 ml (20°C) also soluble in ethanol

Chemical Formula	C <sub>8</sub> H <sub>9</sub> NO <sub>2</sub>
Molecular Weight	151.17
Metabolism	hepatic
Elimination Half Life	1–4 hours

However, there are important differences between the effects of aspirin and those of paracetamol. Prostaglandins participate in the inflammatory response, but paracetamol has no appreciable anti-inflammatory action. Furthermore, COX also produces thromboxanes, which aid in blood clotting — aspirin reduces blood clotting, but paracetamol does not. Finally, aspirin and the other NSAIDs commonly have detrimental effects on the stomach lining, where prostaglandins serve a protective role, but paracetamol is safe.

Indeed, while aspirin acts as an irreversible inhibitor of COX and directly blocks the enzyme's active site, Boutaud *et al.* (2002) found that paracetamol indirectly blocks COX, and that this blockade is ineffective in the presence of peroxides. This might explain why paracetamol is effective in the central nervous system and in endothelial cells but not in platelets and immune cells which have high levels of peroxides.

# FEVERFEW- Mechanism of action

- The pharmacology of feverfew is complex; over 35 chemical constituents of *Tanacetum parthenium* have been identified.
- The best-characterized compounds are contained in the feverfew leaf.
- Feverfew leaves contain 3 different types of sesquiterpene lactones: the germacranolides, the eudesmanolides, and the guaianolides. Parthenolide, a germacranolide, is the most abundant sesquiterpene lactone.
- Additional sesquiterpene lactones are found in lesser or trace amounts, but are pharmacologically important. Other non-sesquiterpene constituents of feverfew include borneol, camphor, flavonoids, pyrethrins, and volatile oils (e.g., monoterpenes); some of these are active.
- Melatonin has recently been found during analysis of both fresh feverfew leaves and in commercial preparations; endogenous melatonin levels are reported decreased in chronic migraine sufferers.
- Parthenolide alone does not account for the plant's pharmacologic activity; several other plant species worldwide contain parthenolide but none are reported to have clinical utility against migraine.

# Effects on inflammation

- Feverfew inhibits the synthesis of the eicosanoids (e.g., prostaglandin and leukotriene) irreversibly.
- However, unlike salicylates or NSAIDs, feverfew is not an inhibitor of cyclooxygenase (COX-1 or COX-2).
- Feverfew appears to prevent the release of arachidonic acid from platelets and inhibit the action of phospholipase A2. Dose-dependent inhibition of thromboxane, leukotriene B4, and inflammatory cytokines has been observed *in vitro*.
- It is not clear if feverfew directly blocks the synthesis of thromboxane.
- Feverfew also reportedly inhibits granular secretion from neutrophils, phagocytosis by neutrophils, and mast cell degranulation and histamine release *in vitro*; precise mechanisms have not been determined.
- Combined, these anti-inflammatory effects may account for the traditional history of using feverfew for inflammatory conditions.

## Effects on platelets and serotonin

- Feverfew inhibits platelet aggregation in vitro via mechanisms different than traditional platelet-inhibiting drugs, but exact actions are unclear.
- Feverfew may inhibit platelet aggregation via modification of platelet sulfhydryl groups in vitro, which then disrupt the platelet membrane changes that produce platelet "clumping".
- Feverfew may or may not also reduce the synthesis of thromboxane.
- Feverfew prevents the formation of clot-like platelet aggregations in response to adenosine diphosphate (ADP), collagen and thrombin, a potential indicator of thrombolytic activity. The herb, by reducing platelet aggregation, may also help maintain the integrity of the cerebral vascular endothelial cells.
- The in vivo effect on platelets is less clear. Clinically, one report noted that the platelet aggregation of patients taking chronic feverfew was no different than that of control subjects.
- Feverfew also inhibits the secretion of various substances (e.g., arachadonic acid, and serotonin) from the platelet.
- In migraine pathology, plasma serotonin levels have been shown to increase before a migraine attack and decrease after the attack.
- Inhibition of serotonin release from platelets may be helpful in migraine prevention. The 5 sesquiterpene lactones which inhibit serotonin release are artecanin, canin, 3-beta-hydroxyparthenolide, parthenolide, and secotanapartenolide A. In addition, parthenolide appears to be a weak 5HT-2A receptor antagonist, similar to other migraine-prophylactic therapies.

# Effects on vascular smooth muscle

- Feverfew inhibits spasm of vascular smooth muscle by blocking voltage-dependent potassium channels; calcium-dependent potassium channels are not affected.
- The spasmolytic activity of feverfew may reduce the reactivity of the cerebral blood vessels to endogenous vasoconstrictive or vasodilatory compounds like norepinephrine, acetylcholine, bradykinins, prostaglandins, histamine and serotonin.

## Other effects

Parthenolide does not exhibit activity against gram-negative organisms. Parthenolide exhibits cytotoxic activity, inhibiting the proper replication of DNA in certain *in vitro* malignant cell lines. More research is needed; mammalian studies have not been conducted.

# Ibuprofen-Mechanism of Action

- Ibuprofen works in the same way as aspirin. It inhibits prostaglandin production so reduces pain and swelling. It belongs to the same family of drugs as aspirin, non-steroidal anti inflammatory drugs (NSAIDs). Like aspirin, ibuprofen sticks to the enzyme, cyclooxygenase, so it can't produce prostaglandins.

## Side Effects

- It has similar side effects to aspirin.
- Most common are rashes, ringing in the ears, headaches, dizziness, drowsiness, abdominal pain, nausea, diarrhea, constipation and heartburn.



# Cocaine



- From the plant called *Erythroxylon coca*, cocaine is a local anesthetic and central nervous system stimulant. It can be taken by chewing on coca leaves, smoked, inhaled ("snorted") or injected.
- A medical account of the coca plant was published in 1569. In 1860, Albert Neiman isolated cocaine from the coca leaf and described the anesthetic action of the drug when it was put on his tongue. Angelo Mariani, in the early 1880s produced a "medicinal" wine, called Vin Mariani, that contained 11% alcohol and 6.5 mg of cocaine in every ounce. The famous psychotherapist, Sigmund Freud, in 1884, recommended cocaine for a variety of illnesses and for alcohol and morphine addictions. Unfortunately, many of his patients went on to become addicted to cocaine! In 1886, John Pemberton developed Coca Cola, a drink that contained cocaine and caffeine. Cocaine was REMOVED from Coca Cola in 1906 (but it still has the caffeine). The Harrison Narcotic Act in 1914 made cocaine illegal. Finally, in 1985, crack cocaine was introduced and rapidly became a major drug problem.

# Opium

- Opium is a narcotic analgesic drug which is obtained from the unripe seed pods of the opium poppy (*Papaver somniferum* L. or the synonym *paeoniflorum*). Opium has powerful narcotic properties. Its constituents and derivatives are used as painkillers in extreme circumstances, such as in terminal stages of cancer. Therefore, a small amount of legal production is discreetly conducted under strict supervision by law enforcement.
- Raw opium has to be processed to produce a form of opium that can be smoked. This form of opium has a considerably higher morphine content percentage-wise than the raw latex. This is then pressed into bricks and either transported to heroin laboratories or used as is.
- Although opium is used in the form of paregoric to treat diarrhea, most opium imported into the United States is broken down into its alkaloid constituents. These alkaloids are divided into two distinct chemical classes, phenanthrenes and isoquinolines. The principal phenanthrenes are morphine, codeine, and thebaine, while the isoquinolines have no significant central nervous system effects and are not regulated under the Controlled Substances act. Opium is also processed into heroin, and most current drug use occurs with processed derivatives rather than with raw opium.



# Chemical properties and hysiological effects

- Opium resin contains two groups of alkaloids : phenanthrenes (including morphine and codeine) and benzyloquinolines (including papaverine). Morphine is by far the most prevalent and important alkaloid in opium, consisting of 10%-16% of the total. It binds to and activates  $\mu$ -opioid receptors in the brain, spinal cord, stomach and intestine. Regular use, even for a few days, invariably leads to physical tolerance and dependence. Various degrees of psychological addiction can occur, though this is relatively rare when opioids are properly used -- for treatment of pain, rather than for euphoric effects. These mechanisms result from changes in nervous system receptors in response to the drug. In response to the drug, the brain creates new receptors for opiates. These receptors are "pseudo" receptors and do not work. When the opiates are out of the body, the brain has more receptors than before the use of the drug, but only the same amount of endogenous opiate (endorphins) to fill these receptors

## Medical Uses

- Opium has been a major item of trade for centuries, and has long been used as a painkiller and sedative. It was well known to the ancient Greeks, who named it **opion** ("poppy juice"), from which the present name—a Latinisation—is derived. Many patent medicines of the 19<sup>th</sup> century were based around laudanum (known as "tincture of opium", a solution of opium in ethyl alcohol). Tincture of opium is prescribed in modern times, among other reasons, for ongoing, severe diarrhea caused, for example, by the creation of an ileostomy. A 10% tincture of opium solution (10% opium, 90% ethyl alcohol) taken 30 minutes prior to meals will significantly slow intestinal motility, giving the intestines greater time to absorb fluid in the stool.

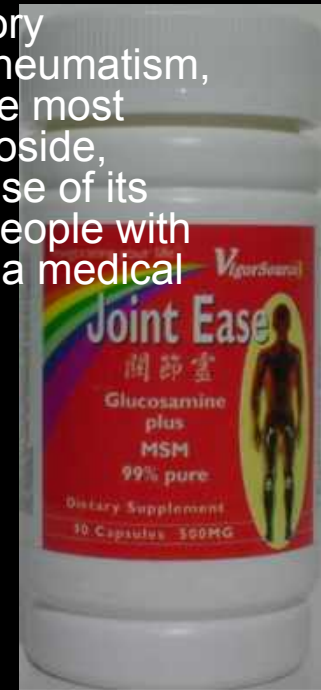
# Laudanum



- Laudanum is an opium tincture, sometimes sweetened with sugar and also called *wine of opium*.
- In the 16<sup>th</sup> century, a Swiss physician named Paracelsus (1493–1541) experimented with the medical value of opium. He decided that its medical (analgesic) value was of such magnitude that he called it Laudanum, from the Latin *laudare*, to praise, or from *labdanum*, the term for a plant extract. He did not know of its addictive properties.
- In the 19<sup>th</sup> century, laudanum was used in many patent medicines to "relieve pain... to produce sleep... to allay irritation... to check excessive secretions... to support the system... [and] as a sudorific". The lack of any genuine treatments meant that opium derivatives were one of the few substances that had any effect, and so laudanum was prescribed for ailments from colds to meningitis to cardiac diseases, in both adults and children. Innumerable Victorian women were prescribed the drug for relief of menstrual cramps and vague aches,
- Laudanum is classified as a Schedule II drug under the Controlled Substances Act. Its most common formulation is known as 'deodorized tincture of opium,' and is manufactured in the United States by Ranbaxy Pharmaceuticals. The only medically-approved uses for laudanum in the United States are for treating diarrhea and pain. Laudanum (deodorized opium tincture) contains the equivalent of 10 milligrams of morphine per milliliter. By contrast, laudanum's weaker cousin, paregoric, is 1/25<sup>th</sup> the strength of laudanum, containing only 0.4 milligrams of morphine per milliliter.

# JointEase Plus

- For those who suffer from arthritis and other muscular skeletal problems.
- JointEase Plus contains 100% pure Harpagophytum Procumbens, also known as 'Sengaparile', 'Devil's Claw' or 'Duiwelsklou', because of the claw-like shape of its fruit. For thousands of years, the Khoisan people of the Kalahari Desert (in Southern Africa) have used Devil's Claw to treat painful joint conditions and other health problems.
- Harpagophytum Procumbens (Devil's Claw): This herb is indigenous to the Kalahari Desert and is exclusive to Africa. Because of its powerful anti-inflammatory properties, Devil's Claw is used world-wide for osteo-arthritis, fibrositis, rheumatism, small joint disease and lower backache. Scientific analysis shows that the most important active ingredients in Devil's Claw include monoterpine, harpagoside, glycoside, beta-sitosterol, procumbine and stigmasterol. Warning: Because of its strong anti-inflammatory properties, JointEase is not recommended for people with stomach ulcers or those with any heart conditions, unless supervised by a medical practitioner.



# MiGone Plus

- Tanacetum parthenium (Feverfew/antifebrin) is a well-known medicinal herb and one of the most widely respected in the prophylactic (preventative) treatment of migraine and chronic headache. There are many clinical studies to support its effectiveness in significantly reducing or completely eliminating the occurrence and the severity of chronic headache and migraine.
- Scientific research has demonstrated that Feverfew contains a range of compounds called sesquiterpene lactones, the principle ingredient being parthenolide. Parthenolide has been scientifically shown to prevent excessive clumping of blood platelets, (but causes blood thinning, therefore high risk of internal bleeding) and to reduce the release of certain pain inducing chemicals and inflammatory compounds.

## Other herbal plants

- Bissy Nut - (Cola acuminata) has been known to help relieve inflammation in disorders such as rheumatism and gout. It also is used as a diuretic, and contains metabolism-enhancing properties.

# TABLE 1

## Side Effects of Select Herbal Products

### Herbal product

Ginkgo biloba

St. John's wort

Ephedra (ma huang)

Kava

### Side effects

Bleeding

Gastrointestinal disturbances, allergic reactions, fatigue, dizziness, confusion, dry mouth, photosensitivity

Hypertension, insomnia, arrhythmia, nervousness, tremor, headache, seizure, cerebrovascular event, myocardial infarction, kidney stones

Sedation, oral and lingual dyskinesia, torticollis, oculogyric crisis, exacerbation of Parkinson's disease, painful twisting movements of the trunk, rash

# TABLE 2

## Drug Interactions with Herbal Products

### Herbal product

Ginkgo biloba

St. John's wort

Ephedra

Ginseng

Kava

### Interacting drugs

Aspirin, warfarin (Coumadin), ticlopidine (Ticlid), clopidogrel (Plavix), dipyridamole (Persantine)

Antidepressants

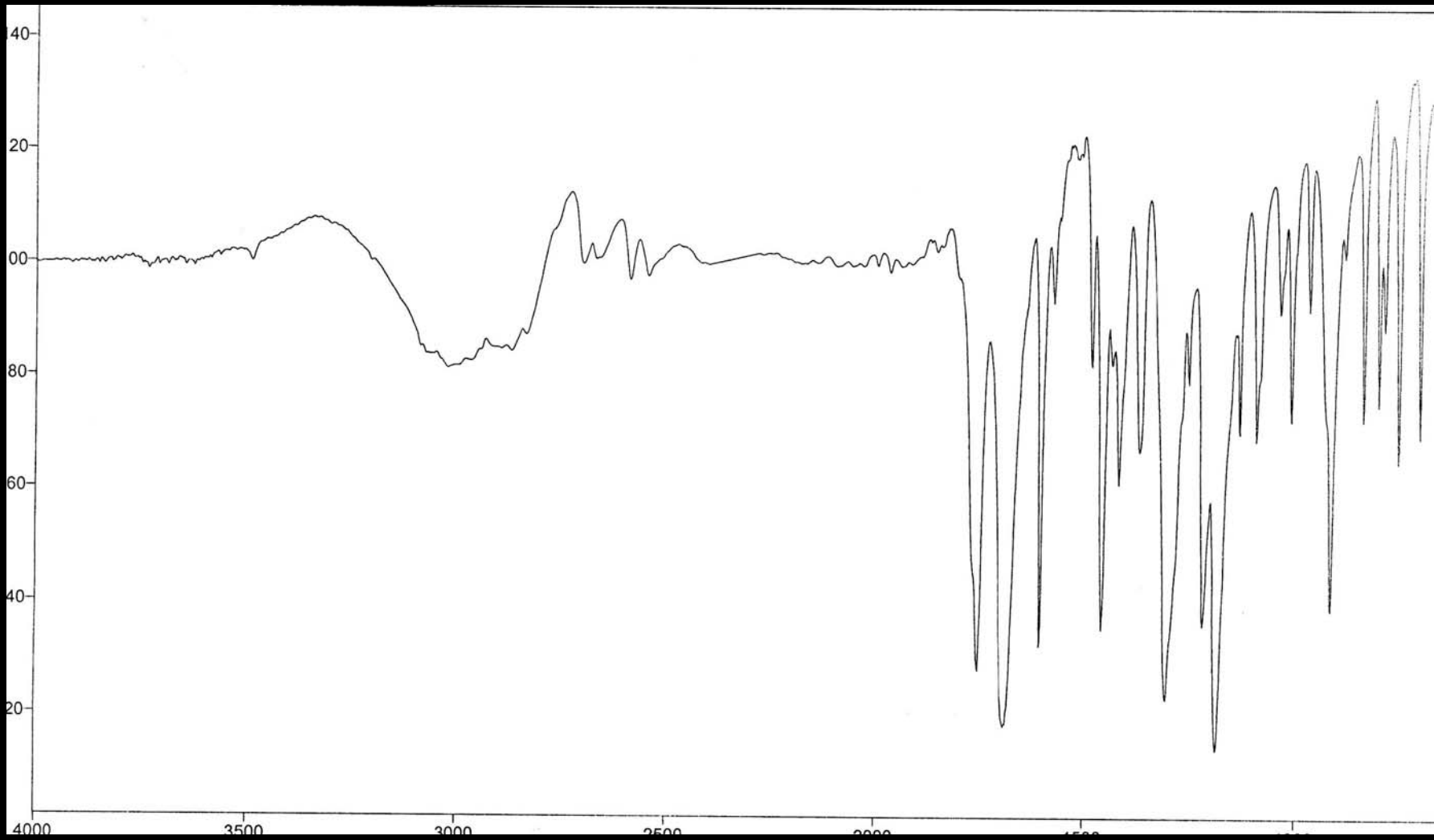
Caffeine, decongestants, stimulants

Warfarin (affects heart rate – stops it)

Sedatives, sleeping pills, antipsychotics, alcohol

**BRADFORD TRIP**

- Salina Parvez (Manager)
- Waqas Ali (Deputy)
- Jawaad Hussain
- Javid Hussain
- Tahera Alam
- Sara O'meara
- Shazna Begum
- Hafeez Mohammed
- Luke Bridgestock
- Nurjahan Begum
- Anam Altaf
- Tahera Begum
- Samehra Parveen



# Infrared Spectroscopy

- Different bonds in compounds absorb different frequencies in infrared spectroscopy.
- In Aspirin there is an ester group and a carboxylic acid group. The carboxylic acid has a C = O and an H-O bond. From our infrared spectroscopy there is a strong peak between 1680-1750 which is due to C = O bond absorption. There is a broad absorption between 2500-3100 which is from the O-H bond from the carboxylic acid group. There is a strong absorption between 1050-1150 this shows the presence of a C-O bond from the ester group. Below 1500 is the fingerprint region which is unique for each compound, in this spectrum the fingerprint region is due to the Benzene and CH<sub>3</sub> (alkyl group).

EI +VE +LMR BSCAN (EXP) UP LR NRM

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Inlet :

119.9

Inten : 9257028

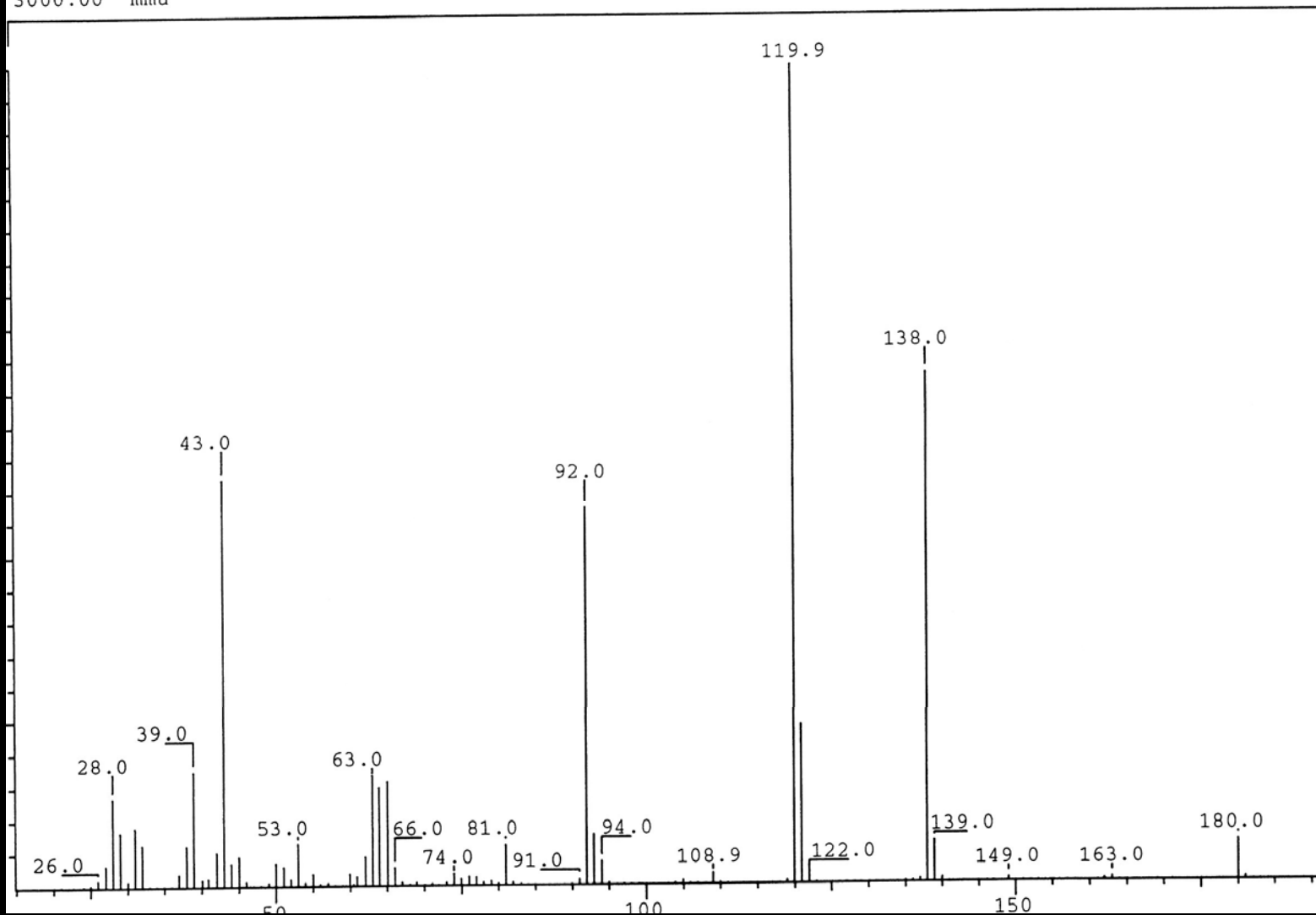
Masses: 20 > 300

119.9

RIC : 41117896

#peaks: 226

3000.00 mmu



# Mass Spectroscopy

Above is a diagram of a mass spectrometer which is used to find the molecular mass of compounds.

There are four stages:

Ionisation – compounds are ionised and are in gaseous state.

Acceleration – compounds pick up speed due to the magnetic field.

Deflection – magnetic field is increased and the compounds separate by their weight.

Detection – the compounds molecular mass is detected.

The molecular mass ( $M_r$ ) of Aspirin is

$$\begin{aligned} C_9O_4H_3 &= (12 \times 9) + (16 \times 4) + (1 \times 3) \\ &= \underline{180} \text{ Mr of Aspirin} \end{aligned}$$

There are 6 peaks on our mass spectrum, which are due to fragmentation:

$M_r = 138$  this peak shows  $C_7H_4O_2$

The  $C_2H_3O$  is broken off.

$M_r = 119.9$  this peak shows  $C_7H_4O_2$

The  $CO_2H$  AND  $CH_3$  are broken off.

$M_r = 92$  this peak shows  $C_6H_4O$

The  $C_2H_3O$  and  $CO_2H$  are broken off.

$M_r = 43$  this peak shows  $C_2H_3O$

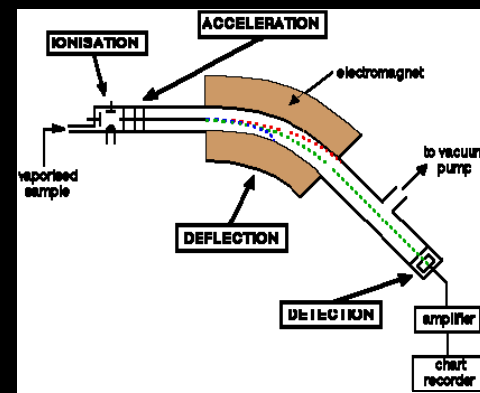
The  $C_6O_2H_5$  is broken off.

$M_r = 63$  this peak shows  $C_3H_4O_3$

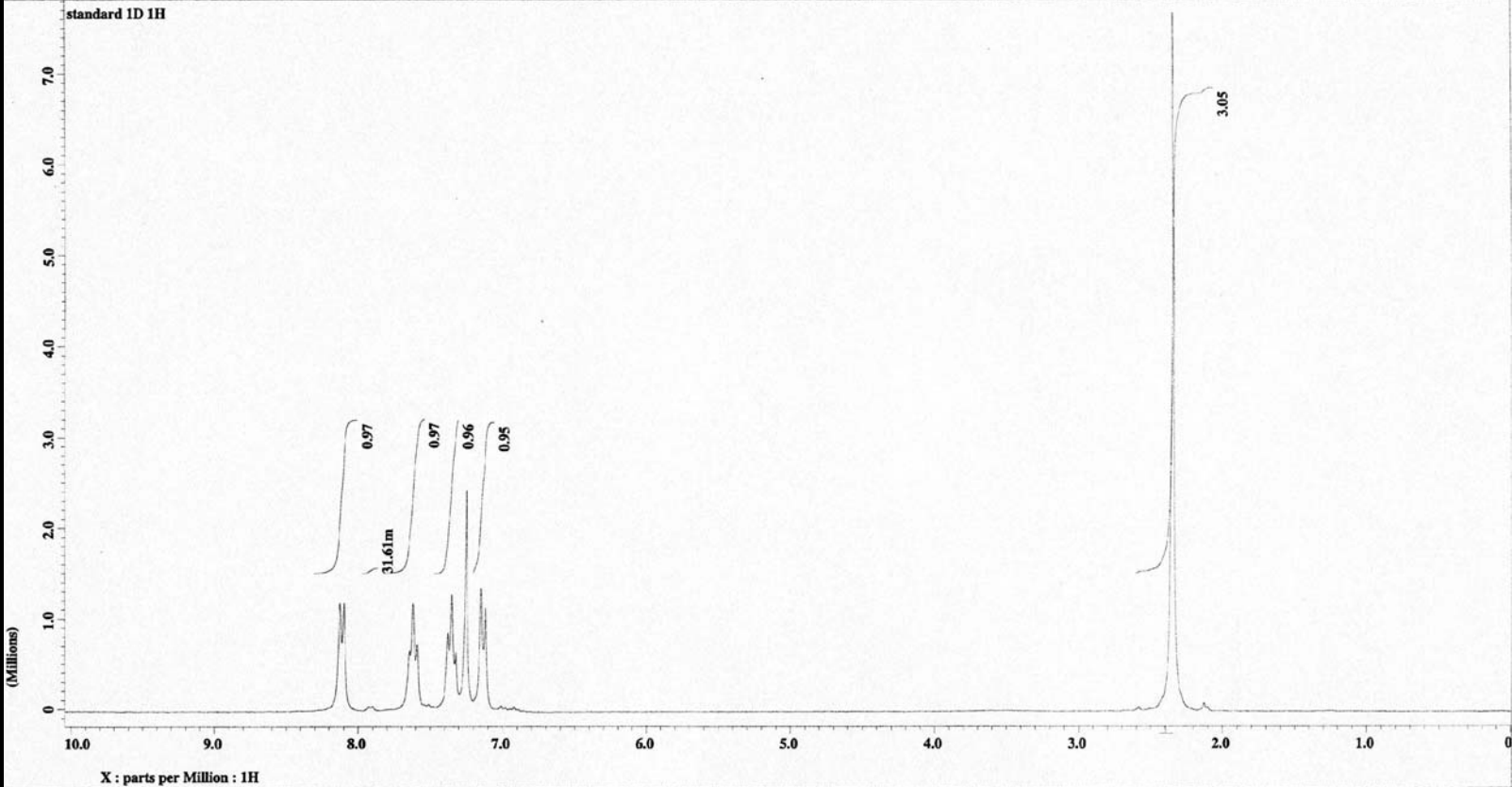
The  $C_6H_4O$  is broken off.

$M_r = 180$  this peak shows the  $M_r$  of Aspirin

$$\begin{aligned} C_9O_4H_3 &= (12 \times 9) + (16 \times 4) + (1 \times 3) \\ &= \underline{180} \text{ Mr of Aspirin} \end{aligned}$$



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Solvent	= CHLOROFORM-D	X_offset	= 5[ppm]	Temp_get	= 16.2[degC]
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Revision_time	= 14-DEC-2005 15:41:25	X_prescans	= 0		
Current_time	= 14-DEC-2005 15:41:51	X_resolution	= 0.12370319[Hz]		
		X_sweep	= 4.05350628[kHz]		
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Data_format	= 1D COMPLEX	Mod_return	= 1		
Dim_size	= 32768	Scans	= 16		
Dim_title	= 1H	Total_scans	= 16		
Dim_units	= [ppm]				
Dimensions	= X	X_90_width	= 12.2[us]		
Site	= GX270 Delta	X_acq_time	= 8.0838656[s]		
Spectrometer	= DELTA_NMR	X_angle	= 45[deg]		
		X_pulse	= 6.1[us]		



# Nuclear Magnetic Resonance Spectroscopy

- Nuclear Magnetic Resonance Spectroscopy shows the number of hydrogens in a compound, the number of different proton environments, number of protons on adjacent carbon, and the ratio of protons in their environments.
- The NMR Spectroscopy there is a huge peak at about 2.4, this chemical shift value proves that there is an alkyl group (CH<sub>3</sub>) attached to a carbonyl group. The peak is all together, as a singlet which shows that there are no carbons on the adjacent carbon using the  $n+1=1$  rule.  $N+1=1$ .  $N=0$ . This indicates that this CH<sub>3</sub> is from the alkyl group on the carbonyl group in the Aspirin. The number on the top of the peak shows that there are 3 hydrogen atoms in this chemical environment.
- There are more peaks between 7-8.2 these are from the Arene group which is Benzene. There are 3 chemical environments on the Benzene therefore the peaks occur showing the presence of Hydrogen (protons) on the Benzene.

# Student Comments

- **Waqas Ali (Deputy)** – ‘It was really remarkable day as we would never have done any of the tasks we did in aim higher pharmacy and thanks to Chris’s good connections at Bradford University we had an excellent day of fun and learning.’
- **Jawaad Hussain** – ‘ I got to see the equipment used to determine mass, IR and NMR spec’
- **Javid Hussain** – ‘ Interesting and fun.’
- **Sara O’meara** – ‘ It was very interesting and useful, and help me understand mass spec.’
- **Shazna Begum** – ‘It helped me with my current chemistry course’
- **Hafeez Mohammed** – ‘ A hands on experience which enabled me to obtain valuable insight in to the various techniques of spec’
- **Nurjahan Begum** - 'it was very useful and interesting!
- **Tahera Begum** – ‘the trip to Bradford University was very interesting, i was surprised at how much the equipments such as NMR cost! it was a very enjoyable’
- **Samehra Parveen** – ‘I enjoyed my visit to Bradford, I am considering going to study pharmacy at Bradford’